2011 Imaging Criteria

Magnetic Resonance Imaging (MRI), Brain[1*RIN, 2, 3, 4, 5]

CLIENT:	Name	D.O.B.	ID#	GROUP#
CPT/ICD9:	Code	Facility	Service Date	
PROVIDER:	Name		ID#	Phone#
	Signature		Date	

ICD-9-CM: 88.91

INDICATIONS (choose one and see below)

☐ 100 Acute onset persistent neurologic Sx/findings (suspected stroke/CVA) ♦
☐ 200 Follow-up study post stroke/CVA
☐ 300 New transient neurologic Sx/findings (suspected TIA) ♦
☐ 400 Headache
☐ 500 Seizure
☐ 600 Head trauma ♦
☐ 700 CNS infection (gadolinium contrast recommended)
☐ 800 Follow-up of intracranial abscess (gadolinium contrast recommended)
☐ 900 Follow-up of primary brain tumor (gadolinium contrast recommended)
☐ 1000 Single brain tumor by CT (gadolinium contrast recommended)
☐ 1100 CNS evaluation for brain metastases (gadolinium contrast recommended)
☐ 1200 Follow-up of AVM
☐ 1300 Post intracranial procedure/craniotomy/craniectomy
☐ 1400 Suspected CNS involvement with systemic disease
☐ 1500 Multiple sclerosis (MS)
☐ 1600 Acoustic neuroma/cerebellar pontine angle tumor (gadolinium contrast recommended)
☐ 1700 Vestibular neuronitis
☐ 1800 Nonacute onset mental status changes/dementia
☐ 1900 Suspected cerebral venous thrombosis
☐ 2000 Hydrocephalus
☐ 2100 Movement disorder
☐ 2200 Preoperative assessment stereotactic introduction, subcortical electrodes/stereotactic lesion creation
☐ 2300 Suspected subdural hematoma (SDH)
☐ Indication Not Listed (Provide clinical justification below)

☐ 100 Acute onset persistent neurologic Sx/findings (suspected stroke/CVA) **[One]** ♦ [6*RIN, 7, 8, 9]
  ☐ 110 Sensory deficit[10]

- ☐ 120 Motor deficit[11, 12]
- ☐ 130 Language deficit[13]
- ☐ 140 Cognitive dysfunction of unknown etiology[14, 15]
- ☐ 150 Visual impairment[16]
- ☐ 160 Altered level of consciousness[17]
- ☐ 170 Vertigo with headache/central nystagmus[18, 19, 20, 21]

☐ 200 Follow-up study post stroke/CVA **[One]**
- ☐ 210 Anticoagulation planned[22]
- ☐ 220 New/worsening CNS Sx/findings ♦[23]

☐ 300 New transient neurologic Sx/findings (suspected TIA) **[One]** ♦[24, 25, 26, 27]
- ☐ 310 Sensory deficit[10]
- ☐ 320 Motor deficit[11, 12]
- ☐ 330 Language deficit[13]
- ☐ 340 Cognitive dysfunction of unknown etiology[14, 15]
- ☐ 350 Visual impairment[16]
- ☐ 360 Vertigo with headache/central nystagmus[18, 19, 20, 21]

☐ 400 Headache **[One]**[28*RIN]
- ☐ 410 Papilledema by PE[29]
- ☐ 420 New headache **[One]**[30]
  - ☐ 421 Age ≥ 50 and no Hx of headaches[31]
  - ☐ 422 Focal neurologic finding by PE[32]
  - ☐ 423 Headache with syncope by Hx[33]
  - ☐ 424 Mental status changes by Hx/PE[34]
  - ☐ 425 Absent venous pulsations by funduscopic exam[35]
  - ☐ 426 Headache onset with exertion/Valsalva maneuver by Hx
  - ☐ 427 Headache causes awakening from sleep by Hx[36]
  - ☐ 428 Headache with nocturnal vomiting by Hx[37]
- ☐ 430 Chronic headache **[One]**[38, 39]
  - ☐ 431 Focal neurologic finding by PE[32]
  - ☐ 432 Headache with syncope by Hx[33]
  - ☐ 433 Mental status changes by Hx/PE[34]
  - ☐ 434 Worsening of previously stable chronic headache by Hx[40]

☐ 500 Seizure **[One]**[41, 42]
- ☐ 510 New onset seizure ♦[43]
- ☐ 520 Refractory to Rx **[All]**[44]

- ☐ 521 Increased seizure activity with therapeutic blood levels of anticonvulsant
- ☐ 522 ≥ 12 wks since initiation of anticonvulsant Rx[45]
- ☐ 523 No concurrent seizure-provoking medications[46]

☐ 600 Head trauma **[Both]** ♦[47]
- ☐ 610 CT not feasible/nondiagnostic for etiology of Sx/findings[48]
- ☐ 620 Sx/findings **[One]**
  - ☐ 621 Retrograde/anterograde amnesia[49, 50]
  - ☐ 622 LOC by Hx/PE
  - ☐ 623 Mental status changes by Hx/PE[34]
  - ☐ 624 Vomiting[51]
  - ☐ 625 Focal neurologic finding by PE[32]
  - ☐ 626 Headache by Hx
  - ☐ 627 Seizure by Hx/PE
  - ☐ 628 Coagulopathy by Hx
  - ☐ 629 Skull fracture by PE/x-ray

☐ 700 CNS infection (gadolinium contrast recommended) **[One]**[52]
- ☐ 710 Suspected infection in immunocompetent host **[Both]** ♦[53]
  - ☐ 711 New/worsening CNS Sx/findings **[One]**[54]
    - ☐ -1 Focal neurologic finding by PE[32]
    - ☐ -2 Headache by Hx
    - ☐ -3 Photophobia[55]
    - ☐ -4 Meningismus[56, 57]
    - ☐ -5 Mental status changes by Hx/PE[34]
    - ☐ -6 Seizure by Hx/PE
  - ☐ 712 Associated findings **[One]**[54]
    - ☐ -1 Temperature > 100.4 F(38.0 C)
    - ☐ -2 WBC > 12,000/cu.mm($12x10^9$/L)
- ☐ 720 Suspected infection in immunocompromised host **[One]** ♦[58, 59]
  - ☐ 721 Focal neurologic finding by PE[32]
  - ☐ 722 Atypical headache by Hx[60]
  - ☐ 723 Mental status changes by Hx/PE[34]
  - ☐ 724 Seizure by Hx/PE
- ☐ 730 Follow-up assessment[61]

☐ 800 Follow-up of intracranial abscess (gadolinium contrast recommended) **[One]**[62, 63]
- ☐ 810 New/worsening CNS Sx/findings **[One]** ♦
  - ☐ 811 Focal neurologic finding by PE[32]

- ☐ 812 Vomiting
- ☐ 813 Headache by Hx
- ☐ 814 Mental status changes by Hx/PE[34]
- ☐ 815 Seizure by Hx/PE

☐ 820 Follow-up assessment during Rx[64]

☐ 830 Follow-up assessment after Rx completed

☐ 900 Follow-up of primary brain tumor (gadolinium contrast recommended) **[One]**[65*RIN, 66, 67, 68]

☐ 910 New/worsening CNS Sx/findings ♦

☐ 920 Periodic assessment[69]

☐ 1000 Single brain tumor by CT (gadolinium contrast recommended)[67]

☐ 1100 CNS evaluation for brain metastases (gadolinium contrast recommended) **[One]**[70]

☐ 1110 Baseline scan as part of staging **[One]**[71*MDR]

- ☐ 1111 Sarcoma
- ☐ 1112 Melanoma[72, 73]
- ☐ 1113 Small cell lung cancer[74]

☐ 1120 Baseline scan positive **[One]**[75]

- ☐ 1121 Periodic assessment during chemotherapy/radiation Rx[76]
- ☐ 1122 Restaging after chemotherapy/radiation Rx completed

☐ 1130 New/worsening CNS Sx/findings **[One]** ♦[77]

- ☐ 1131 Known cancer elsewhere
- ☐ 1132 Known brain metastasis by prior CT/MRI

☐ 1200 Follow-up of AVM[78*RIN, 79, 80, 81]

☐ 1300 Post intracranial procedure/craniotomy/craniectomy **[One]**[82]

☐ 1310 New/worsening CNS Sx/findings ♦

☐ 1320 Follow-up assessment[83]

☐ 1400 Suspected CNS involvement with systemic disease **[One]**

☐ 1410 Systemic lupus erythematosus (SLE)/vasculitis[84, 85]

☐ 1420 HIV[82]

☐ 1430 Sarcoidosis (gadolinium contrast recommended)[86]

☐ 1500 Multiple sclerosis (MS) **[One]**[87, 88, 89]

☐ 1510 Suspected MS **[One]**[90, 91, 92, 93]

- ☐ 1511 Clinically isolated syndrome (gadolinium contrast recommended) **[One]**[94]

- ☐ -1 Optic neuritis[95, 96]
- ☐ -2 Ophthalmoplegia[97]
- ☐ -3 Transverse myelitis[98, 99]

☐ 1512 CNS deficit not in dermatomal/peripheral nerve distribution and other etiologies excluded **[One]**

- ☐ -1 Sensory deficit
- ☐ -2 Motor dysfunction

☐ 1513 Loss of coordination and other etiologies excluded

☐ 1520 Known MS with new/worsening symptoms (gadolinium contrast recommended)[100*MDR]

☐ 1600 Acoustic neuroma/cerebellar pontine angle tumor (gadolinium contrast recommended) **[One]**[101, 102]

☐ 1610 Suspected acoustic neuroma/cerebellar pontine angle tumor **[Both]**

- ☐ 1611 Unilateral hearing loss/tinnitus with ear normal by PE[103]
- ☐ 1612 Findings **[One]**[104]
  - ☐ -1 Asymmetric neural hearing loss by audiometry[105]
  - ☐ -2 Abnormal acoustic reflex testing[106]
  - ☐ -3 Roll-over by phonetically balanced word testing[106]

☐ 1620 Follow-up known acoustic neuroma **[One]**

- ☐ 1621 6 mos from diagnosis/annual follow-up[107]
- ☐ 1622 Post radiosurgery/surgical excision[108]

☐ 1700 Vestibular neuronitis **[All]**[109*RIN, 110]

☐ 1710 Vertigo **with** associated Sx/findings **[One]**[18]

- ☐ 1711 Nausea/vomiting
- ☐ 1712 Nystagmus[19, 111]
- ☐ 1713 Postural instability

☐ 1720 Ear normal by PE

☐ 1730 Continued/worsening vertigo **after** Rx **[Two]**[112, 113]

- ☐ 1731 Antihistamine Rx ≥ 1 wk[114]
- ☐ 1732 Neuroleptic Rx ≥ 1 wk[115]
- ☐ 1733 Benzodiazepine Rx ≥ 1 wk[116]
- ☐ 1734 Anticholinergic Rx ≥ 1 wk[117]
- ☐ 1735 Hydroxyzine Rx ≥ 1 wk

☐ 1800 Nonacute onset mental status changes/dementia **[All]**[118, 119, 120]

☐ 1810 Sx/findings **[One]**[121]

- ☐ 1811 Memory loss by Hx/PE[122]
- ☐ 1812 Confusion/disorientation by Hx/PE[123]

- ☐ 1813 Behavioral disturbance by Hx/PE[124]
- ☐ 1814 Deterioration in intellectual function by Hx/PE

☐ 1820 Depression screening completed[125]

☐ 1830 Lab results nondiagnostic for etiology of mental status change **[All]**

- ☐ 1831 Na >128 mEq/L(128 mmol/L)
- ☐ 1832 Glucose > 60 and < 400 mg/dL(3.33 and < 22.20 mmol/L)
- ☐ 1833 BUN < 80 mg/dL(28.6 mmol/L)
- ☐ 1834 Ca < 11 mg/dL(2.75 mmol/L)
- ☐ 1835 TSH normal[126]
- ☐ 1836 LFTs/ammonia normal[127]
- ☐ 1837 B_{12} normal
- ☐ 1838 RPR negative/not indicated

☐ 1840 Urine drug/toxicology screen **[One]**[128]

- ☐ 1841 Negative
- ☐ 1842 Not indicated

☐ 1900 Suspected cerebral venous thrombosis **[Both]**[129, 130]

☐ 1910 Headache **with** associated Sx/findings **[One]**

- ☐ 1911 Papilledema by PE[29]
- ☐ 1912 Focal neurologic finding by PE[32]
- ☐ 1913 Mental status changes by Hx/PE[34]
- ☐ 1914 Seizure by Hx/PE

☐ 1920 Finding **[One]**

- ☐ 1921 Hypercoagulable state[131]
- ☐ 1922 Skull fracture over dural sinus
- ☐ 1923 Calvarial mass[132]
- ☐ 1924 Infection **[One]**
  - ☐ -1 Rhinosinusitis
  - ☐ -2 Otitis media

☐ 2000 Hydrocephalus **[One]**

☐ 2010 Suspected normal pressure hydrocephalus **[Two]**[133, 134, 135]

- ☐ 2011 Urinary incontinence
- ☐ 2012 Apraxic gait[136]
- ☐ 2013 New onset dementia

☐ 2020 Normal pressure hydrocephalus by Hx with new/worsening CNS Sx/findings ♦[137]

☐ 2030 Suspected obstructive hydrocephalus **[Both]**[138]

- ☐ 2031 Sx/findings **[One]**
  - ☐ -1 Headache by Hx

- ☐ -2 Mental status changes by Hx/PE[34]
- ☐ -3 Papilledema by PE[29]
- ☐ -4 Impaired coordination/ataxia by PE[139, 140]
- ☐ -5 Focal neurologic finding by PE[32]
- ☐ -6 Seizure by Hx/PE

☐ 2032 Risk factor **[One]**

- ☐ -1 AVM/aneurysm by Hx[79]
- ☐ -2 SAH/intraventricular hemorrhage by Hx
- ☐ -3 Meningitis
- ☐ -4 Hydrocephalus by Hx

☐ 2100 Movement disorder **[One]**

- ☐ 2110 Suspected Huntington's chorea and genetic testing not feasible/refused[141, 142, 143]
- ☐ 2120 Progressive ataxia of undetermined etiology[139, 144]

☐ 2200 Preoperative assessment stereotactic introduction, subcortical electrodes/stereotactic lesion creation[145]

☐ 2300 Suspected subdural hematoma (SDH) **[All]**[146]

- ☐ 2310 Sx/findings **[One]**
  - ☐ 2311 LOC by Hx/PE
  - ☐ 2312 Mental status changes by Hx/PE[34]
  - ☐ 2313 Bradyphrenia/apathy by Hx/PE[147]
  - ☐ 2314 Focal neurologic finding by PE[32]
  - ☐ 2315 New onset/worsening headache **with** associated Sx/findings **[One]**
    - ☐ -1 Persistent vomiting by Hx
    - ☐ -2 Pupillary changes[148]
  - ☐ 2316 Seizure by Hx/PE
  - ☐ 2317 Syncope by Hx[33]
  - ☐ 2318 Skull fracture by PE/x-ray
- ☐ 2320 Risk factor **[One]**
  - ☐ 2321 Alcohol abuse by Hx
  - ☐ 2322 Head trauma by Hx/PE
  - ☐ 2323 Anticoagulation Rx
  - ☐ 2324 Coagulopathy
  - ☐ 2325 Age ≥ 65[149]
  - ☐ 2326 CSF shunt by Hx[150]
- ☐ 2330 CT normal/nondiagnostic for subdural hematoma[151]

Notes

(1)-RIN:

For suspected pituitary disease, see the "Magnetic Resonance Imaging (MRI), Pituitary" criteria subset.

(2)

An MRI study using a contrast material such as gadolinium will, in specific circumstances, improve the sensitivity of the study. Gadolinium causes highly vascular tissue and areas of disrupted blood-brain barrier to appear brighter on MRI.

(3)

Current magnetic resonance techniques lack ionizing radiation and provide images with high spatial resolution, excellent soft-tissue contrast, and multi-planar imaging capability (Widjaja and Raybaud, Neurosurg Focus 2008; 25(3): E3). Newer techniques include diffusion-weighted MRI and magnetic resonance spectroscopy. Diffusion-weighted imaging is being used for the evaluation of demyelinating disorders, presurgical evaluation and planning of brain tumors, and seizure disorders. Magnetic resonance spectroscopy is being used for evaluating many brain disorders, including brain tumors, leukodystrophies, and brain injuries (Abdelhalim and Alberico, Neurol Clin 2009; 27(1): 285-301, x; Provenzale, Emerg Radiol 2007; 14(1): 1-12).

(4)

CT and MRI each have relative advantages and disadvantages. CT, which tends to be better tolerated by patients, has the advantages of a shorter study time, better sensitivity for detecting acute hemorrhage, and excellent visualization of bony structures with less degradation of image quality by motion artifact. CT is often the preferred modality in rapidly evolving neurologic conditions (e.g., SAH, ICH) because it is widely available and may be performed easily in the setting of life support equipment. CT, however, poses the disadvantage of patient radiation exposure which carries an increased long-term risk of cancer. MRI tends to be more sensitive in detecting lesions in the brain, as well as assessment of cerebral ischemia. In acute stroke imaging, both CT and MRI are used to rapidly obtain the necessary anatomical, vascular, and functional information (Adam and Dixon, Grainger & Allison's Diagnostic Radiology: A Textbook of Medical Imaging 5th ed. 2008, 1936 p.).

(5)

The following are examples of relative and absolute contraindications to the use of magnetic resonance imaging:

- Implanted devices that are electrically or magnetically activated (e.g., cardiac pacemakers, automatic cardioverter defibrillators, drug infusion pumps, cochlear implants)
- Ferromagnetic metal objects (e.g., cerebral aneurysm clips, intraocular metallic foreign body, prostheses, screws)
- Pregnancy, first trimester
- Renal insufficiency in cases when magnetic resonance imaging is performed with gadolinium-based contrast

(6)-RIN:

MRA of the brain may be performed following a stroke to determine etiology as well as appropriateness and timing of treatment. If MRA or CTA of the brain is requested, see the appropriate indication in the "Computed Tomographic Angiogram (CTA), Brain/Magnetic Resonance Angiogram (MRA), Brain" criteria subset.

(7)

The clinical presentation of stroke is typically an abrupt onset of focal neurological signs and symptoms corresponding to a given cerebrovascular territory. History and physical examination remain the pillars of diagnosing stroke.

(8)

The primary goals of acute stroke imaging are to distinguish ischemic stroke from intracerebral hemorrhage, to select ischemic stroke patients for reperfusion therapies, and to exclude conditions that mimic acute cerebral ischemia (Adams et al., Stroke 2007; 38(5): 1655-1711). Both CT and MRI may be used to evaluate acute neurologic changes, however, CT is more readily available. CT has the advantage of a shorter study time, assists in discriminating nonvascular causes of neurologic symptoms (e.g., brain tumor), and distinguishes acute ischemic from hemorrhagic stroke. MRI is valuable in acute stroke patients with unusual presentations, stroke varieties, or when a stroke mimic is suspected but not clearly identified on CT. Newer MRI techniques, such as diffusion weighted imaging (DWI), have increased sensitivity for detecting the presence, size, location, and extent of ischemia (European Stroke Organisation (ESO) Executive Committee, Cerebrovasc Dis 2008; 25(5): 457-507; The National Collaborating Centre for Chronic Conditions, National clinical guideline for diagnosis and initial management of acute stroke and transient ischaemic attack. 2008 [cited 2010 April 3]; Adams et al., Stroke 2007; 38(5): 1655-1711; Kidwell et al., JAMA 2004, 292: 1823-30).

(9)
The exact time of symptom onset is critical for determining the appropriate imaging study to obtain and the eligibility for thrombolysis (Gorelick and Ruland, Dis Mon 2010; 56(2): 39-100; Yew and Cheng, Am Fam Physician 2009; 80(1): 33-40). Patients admitted within 3 hours of the symptom onset of stroke may be candidates for intravenous tPA based on the National Institute of Neurologic Disorders and Stroke (NINDS) trial (Chuang et al., Am J Emerg Med 2003; 21(3): 167-172; NINDS rt-PA Stroke Study Group, N Engl J Med 1995; 333(24): 1581-1587). The European Cooperative Acute Stroke Study (ECASS III) trial demonstrated clinical efficacy if treatment was instituted up to 4.5 hours after the onset of symptoms. The American Heart Association Stroke Council has, therefore, expanded the treatment window for acute ischemic stroke to 4.5 hours based on the results of the ECASS III trial (Bluhmki et al., Lancet Neurol 2009; 8(12): 1095-1102; Del Zoppo et al., Stroke 2009, 40: 2945-8; Hacke et al., N Engl J Med 2008; 359(13): 1317-1329).

(10)
Sensory deficits commonly seen in stroke or TIA include hemianesthesia, single limb anesthesia, facial hemianesthesia or hypesthesia, and contralateral neglect (a neglect of the side opposite to the brain insult).

(11)
Motor deficits include motor weakness or paralysis (hemiparesis, quadriparesis, single limb involvement, or unilateral facial weakness), impaired coordination or ataxia, or dysphagia.

(12)
Motor weakness occurring with a stroke or TIA tends to be focal (e.g., injury to a particular area of the brain results in a specific deficit).

(13)
Language deficits most commonly present as aphasia or dysarthria.

(14)
Cognitive dysfunction may present as memory loss, confusion, disorientation, or behavioral changes.

(15)
Patients presenting with an acute onset of cognitive dysfunction should undergo imaging to search for potentially reversible structural causes of the mental status change, most notably subdural hematoma or acute hemorrhage (Christensen, Am J Emerg Med 2004; 22(3): 228-229). Acute cognitive changes are more likely the result of a toxic or metabolic disorder rather than structural pathology; however, these patients often cannot provide a clear history, making imaging, along with a metabolic evaluation, an important element of the diagnostic algorithm.

(16)
Common visual symptoms include new vision loss, diplopia, visual field impairment, gaze impairment, and amaurosis fugax (transient ipsilateral monocular blindness).

(17)
Altered levels of consciousness (e.g., coma, marked lethargy) may be due to a variety of mechanisms (e.g., increased ICP, infarct involving arousal systems).

(18)-DEF:
Vertigo is a sensation of motion or spinning. Vertigo may be caused by disorders of CN VIII, the inner ear, the brainstem, or the cerebellum.

(19)-DEF:
Nystagmus is a form of involuntary eye movement. It is characterized by slow movement in one direction and a rapid corrective movement in the other direction. The direction of nystagmus has been traditionally defined as the direction of the rapid corrective component. It can be vertical, horizontal or rotatory.

(20)
Vertigo is very common and, in and of itself, is not an indication for CT or MRI. However, the abrupt onset of vertigo associated with neurologic symptoms (e.g., impaired coordination, ataxia, diplopia, dysphagia, dysarthria, motor or sensory deficits) should raise concerns about intracranial hemorrhage, posterior fossa infarct, or TIA (Kerber, Semin Neurol 2009; 29(5): 482-490; Yew and Cheng, Am Fam Physician 2009; 80(1): 33-40).

(21)

Although headaches are common in adults and often benign in nature, headaches associated with vertigo can be the initial symptom of a life-threatening event such as a brainstem or cerebellar stroke. Imaging is warranted for patients presenting with a sudden, severe headache or a progressively worsening headache associated with the acute onset of vertigo.

(22)

Depending on the clinical circumstances, patients who are not candidates for thrombolysis at presentation (e.g., uncontrolled HTN) may be candidates for anticoagulation. In this setting, imaging should be performed to help estimate infarct size, as well as to exclude brain hemorrhage. MRI is the preferred imaging, but CT may also be used (Krishnan et al., J Thromb Thrombolysis 2010; 29(3): 368-377; Albers et al., Chest 2008; 133(6 Suppl): 630S-669S; Adams et al., Stroke 2007; 38(5): 1655-1711).

(23)

In patients with new or worsening CNS symptoms or findings, CT or MRI may be used.

(24)-DEF:

Transient ischemic attack (TIA) is defined as a transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute brain tissue infarction. Clinical symptoms generally last for less than one hour.

(25)

TIA and stroke are both serious conditions that may cause permanent injury to the brain or lead to disability or death. Early treatment with thrombolytics, anticoagulation, antiplatelet therapy, or surgery can halt or reverse permanent brain damage.

(26)

Whether to perform CT or MRI in this setting is a matter of clinical judgment. Both studies can be used to screen for diseases that mimic TIA (e.g., tumor, subdural hematoma). MRI is the preferred study for suspected TIA, as it may detect small lacunar infarcts not well seen on CT and identify new or preexisting ischemic lesions. Because patients with TIA are at a higher risk for recurrent ischemic events, carotid imaging (e.g., US, MRA, CTA) may be warranted to identify an underlying cause (Easton et al., Stroke 2009; 40(6): 2276-2293; European Stroke Organisation (ESO) Executive Committee, Cerebrovasc Dis 2008; 25(5): 457-507; The National Collaborating Centre for Chronic Conditions, National clinical guideline for diagnosis and initial management of acute stroke and transient ischaemic attack. 2008 [cited 2010 April 3]).

(27)

Virtually any transient focal neurologic symptom may reflect a TIA. Some of the more common presentations include weakness of an extremity, localized sensory disturbance, unilateral visual loss, and communication difficulty. Although the acute onset of cognitive dysfunction can occur with a TIA, it is uncommon for patients to present with transient memory loss or confusion without other neurologic symptoms.

(28)-RIN:

These criteria address headache when there is no specific underlying etiology. If there is concern that the headache may be caused by a SAH, see indication 400 in the "Computed Tomography (CT), Brain" criteria subset. If there is concern that the headache may be a sign of a stroke, TIA, or CNS infection, see indication 100, 300, or 700, respectively in this criteria subset.

(29)-DEF:

Papilledema is swelling of the optic disc, manifested by indistinct margins, hyperemia, venous engorgement, and lack of normal venous pulsations. Papilledema is a sign of increased ICP.

(30)

These criteria address a new or not as yet investigated headache. The headache may actually have been present for some time before the patient seeks medical attention. As the incidence of serious intracranial problems is extremely low in patients with a headache and a normal neurologic exam, a selective approach to ordering imaging studies in these patients is appropriate. These criteria define risk factors which are worrisome for intracranial pathology (e.g., tumor, hemorrhage). MRI is generally preferred over CT for the evaluation of headaches. CT is sensitive for identifying SAH or subdural hematoma, but MRI is more sensitive than CT for identifying lesions of the posterior fossa, ischemia, cerebral venous thrombosis, neoplasms, meningeal disease, and cerebral abscess (Edlow et al., Ann Emerg Med 2008; 52(4): 407-436; Karis, AJNR Am J Neuroradiol 2008; 29(6): 1222-1224; Schaefer et al., J Am Coll Radiol 2007; 4(8): 566-569).

(31)

Neuroimaging is appropriate for patient's ≥ 50 years of age presenting with a new headache. Headache in a patient in this age group who has no previous history of headaches may indicate significant disease (e.g., temporal arteritis, mass) (Edlow et al., Ann Emerg

Med 2008; 52(4): 407-436; Karis, AJNR Am J Neuroradiol 2008; 29(6): 1222-1224; Schaefer et al., J Am Coll Radiol 2007; 4(8): 566-569).

(32)
Focal neurologic finding refers to a specific deficit that corresponds to a particular area of the brain (e.g., right arm weakness from a left motor cortex insult).

(33)-DEF:
Syncope is the transient loss of consciousness and postural tone caused by diminished cerebral blood flow characterized by rapid onset, short duration, and spontaneous complete recovery.

(34)
Mental status changes include confusion, lethargy, disorientation, somnolence, stupor, and coma.

(35)
Venous pulsations occur normally due to variation in pressure along the retinal vein. Cessation of venous pulsation occurs with a rise in ICP, making this finding a sensitive marker of increased ICP (Jacks and Miller, J Neurol Neurosurg Psychiatry 2003; 74(1): 7-9).

(36)
Headaches causing patients to awaken from sleep may be caused by increased ICP.

(37)
Nocturnal vomiting may signify an intracranial tumor or may represent increased ICP.

(38)
Migraine is the most common type of chronic headache, triggered by factors such as stress, certain foods, hormonal changes, or lack of sleep. Treatment involves sleep, analgesics, and biobehavioral therapies (e.g., diet, relaxation techniques, cognitive therapies). Diagnosis of most patients with chronic headaches (e.g., migraine, tension headache) usually does not require imaging, particularly in the absence of neurologic signs and symptoms (Friedman and Grosberg, Emerg Med Clin North Am 2009; 27(1); 71-87; Lewis, Neurol Clin 2009; 27(2): 481-501; Pearlman, Prim Care 2004; 31(2): 407-415, viii).

(39)
Imaging is not generally indicated for patients with chronic headache without other associated neurologic signs and symptoms; it is uncommon to discover any imaging abnormalities in this setting (Jordan, AJNR Am J Neuroradiol 2007; 28(9): 1824-1826; McConaghy, Prim Care 2007; 34(1): 83-97). For patients with chronic headache and associated neurologic symptoms, imaging may be indicated. MRI is the study of choice due to its enhanced sensitivity in the detection of intracranial pathology (Schaefer et al., J Am Coll Radiol 2007; 4(8): 566-569).

(40)
Headache patterns of frequency and severity are often cyclical and related to environmental, hormonal, or social factors. Significant worsening of a previously stable headache can indicate new pathology and warrants imaging.

(41)
Seizure disorders may be of varying focality and intensity. Status epilepticus is a condition characterized by seizures lasting for at least 30 minutes that are either continuous or rapidly repeating, such that recovery (return to full consciousness) does not occur between attacks. Generalized or grand mal seizures involve the entire brain and are associated with generalized convulsions and LOC. Partial or focal seizures result from epileptiform activity localized to one area of the brain. The particular area that is affected will determine the resulting symptom. Complex partial or focal seizures, formerly referred to as temporal lobe and psychomotor seizures, are characterized by the episodic loss of conscious "awareness," and may begin as a simple partial seizure. Complex partial seizures may subsequently generalize (to involve the entire brain) and cause total loss of consciousness and convulsions.

(42)
Seizure with accompanying neurologic findings may suggest an anatomic cause for the seizure (Adams and Knowles, Am Fam Physician 2007; 75(9): 1342-1347; Shneker and Fountain, Dis Mon 2003; 49(7): 426-478).

(43)
Neuroimaging is recommended for the evaluation of patients presenting with a first unprovoked seizure (Krumholz et al., Neurology 2007; 69(21): 1996-2007; ACEP Clinical Policies Committee, Ann Emerg Med 2004; 43(5): 605-625). MRI is the study of choice due to its enhanced sensitivity, although CT is a reasonable alternative when MRI is not feasible (National Institute for Clinical Excellence (NICE), The epilepsies. Clinical Guideline 20. 2004, 73; Shneker and Fountain, Dis Mon 2003; 49(7): 426-478).

(44)
Refractory seizures do not require imaging unless a new lesion is suspected (e.g., change in seizure pattern) or management will change based on the results of the scan.

(45)
Time is required to adjust medications and assess their results before assuming that the seizures are refractory to treatment.

(46)
Prescription medications such as TCAs, antipsychotics, theophylline, and lidocaine can lower the seizure threshold. Attempts should be made to reduce or discontinue all such medications, but the risks and benefits of such an intervention need to be considered for each patient.
Recreational CNS stimulants (e.g., cocaine) can also cause seizures.

(47)
After head trauma, CT is the initial imaging procedure of choice to assess for fracture and to exclude intracranial hemorrhage. MRI may be more sensitive in assessing neural injury after the first 24 hours and for detecting small subdural hematomas. With evolving MRI technology, such as magnetic resonance spectroscopy, diffusion-tensor imaging, and magnetization transfer MRI, there may be an increase in future utility of MRI in head trauma imaging (Jagoda et al., Ann Emerg Med 2008; 52(6): 714-748; Sigmund et al., Pediatr Neurol 2007; 36(4): 217-226; Smits et al., Radiology 2007; 245(3):831-838).

(48)
For the evaluation of acute head trauma, CT is preferred to MRI for detecting hemorrhage and possible fracture. If the initial CT is negative but symptoms persist or worsen, MRI is recommended due to its ability to detect subtle areas of brain contusion (Provenzale, Emerg Radiol 2007; 14(1): 1-12).

(49)-DEF:
Retrograde amnesia is having no memory of the events that occurred before the trauma or condition.

(50)-DEF:
Anterograde amnesia is having no memory for events that occurred after the trauma or condition.

(51)
Vomiting after head trauma may be an early indication of increased ICP.

(52)
The superior contrast resolution of MRI makes it a more sensitive imaging tool for evaluating intracerebral abnormalities associated with a variety of complicated CNS infectious processes. Gadolinium contrast improves lesion delineation, localizes regions likely to provide positive biopsy, and identifies active disease. Additional information may be obtained from using diffusion-weighted imaging. In uncomplicated cases, CT may be obtained initially to identify patients at higher risk for herniation with intracranial abnormalities such as hydrocephalus, mass lesions, cerebral edema, and midline brain shift. LP often follows CT in the event the patient is considered low risk for herniation (Fitch et al., Infect Dis Clin North Am 2008; 22(1): 33-52, v-vi; Kastrup et al., NeuroRx 2005; 2(2): 324-332).

(53)
These criteria address CNS infection in the immunocompetent host, where the concern is generally for meningitis or encephalitis.

(54)
In the setting of meningitis or encephalitis, an immunocompetent host will usually demonstrate signs of infection such as fever, elevated WBC, neck stiffness, or neurologic signs. Imaging in this context is helpful to rule out other possible etiologies that might confound or complicate the diagnosis.

(55)-DEF:
Photophobia is abnormal visual intolerance to light.

(56)-DEF:
Meningismus is a symptom complex associated with meningeal irritation, such as neck stiffness or a positive Kernig's or Brudzinski's sign (stretching of the nerve roots causes neck pain).

(57)
Meningismus may be seen with any meningeal irritant but should raise the suspicion of infectious meningitis.

(58)
Immunocompromised hosts are individuals whose immune system is defective either because of a primary underlying immunodeficiency disorder or because of the administration of medications that suppress the immune response.

(59)
Although both CT and MRI are effective imaging tools in patients with AIDS-related disease, MRI is the study of choice. MRI is preferred for tuberculous meningitis, common in patients with AIDS, because it can reveal small infarctions, granulomas, and inflammation of the ventricles. MRI is also preferred in suspected fungal infections due to its superior resolution and sensitivity to tissue edema (Fitch et al., Infect Dis Clin North Am 2008; 22(1): 33-52, v-vi; Kastrup et al., NeuroRx 2005; 2(2): 324-332).

(60)
These criteria address headaches of concern to the provider and patient because of features that are atypical for the patient. The headache may be atypical because it is unusually severe, long-lasting, or because it has new or differing characteristics from prior headaches.

(61)
Follow-up assessment is not necessary more frequently than every 7 to 10 days if the patient is stable or improving.

(62)
Abscesses involving the CNS are uncommon. They sometimes result from direct trauma or neurosurgery, but may be caused by meningitis, rhinosinusitis, mastoiditis, and other extra-cranial sources (Ziai and Lewin, Neurol Clin 2008; 26(2): 427-468, viii).

(63)
Whether to perform a CT or MRI in this setting is a matter of clinical judgment. While MRI provides greater detail, CT is often sufficient for follow-up studies. It is generally best to perform the same study serially as this allows direct comparison of studies.

(64)
The frequency of assessment is a matter of clinical judgment based upon the size, location, and number of intracranial abscesses. Follow-up is necessary to monitor resolution of the abscess in response to antibiotic therapy. If there is no progress, surgical drainage may be necessary.

(65)-RIN:
These criteria address a previously diagnosed brain tumor. For symptomatology which makes one suspect a new brain lesion, see the appropriate indication within this criteria subset.

(66)
The most common primary brain tumors in adults are gliomas and meningiomas, with gliomas accounting for more than 80% of primary brain tumors (Chandana et al., Am Fam Physician 2008; 77(10): 1423-1430; Grant, J Neurol Neurosurg Psychiatry 2004; 75 Suppl 2: ii18-23).

(67)
Gadolinium-enhanced MRI (GdMRI) is the preferred imaging method for evaluating patients with suspected or confirmed primary tumor or metastatic intraspinal extension, suspected or confirmed disc space infection, or an epidural abscess (Chin, Semin Neurol 2002; 22(2): 205-220; Runge et al., Top Magn Reson Imaging 2001; 12(4): 231-263). Contrast improves lesion delineation, localizes regions likely to provide positive biopsy, and identifies active disease (Jacobs et al., NeuroRx 2005; 2(2): 333-347).

(68)
Whether to perform a CT or MRI in this setting is a matter of clinical judgment. MRI is preferred for imaging all types of brain tumors because of its high sensitivity, its ability to identify tumor sites near bone, its sensitivity to tissue edema, and its capability of accurately delineating tumors and their relationship to normal structures.

(69)
Periodic assessment is performed to evaluate response to therapy.

(70)
MRI with contrast is considered the best method for imaging brain metastases. The contrast enhances any disruption of the blood-brain barrier that can occur with tumors (American College of Radiology (ACR), ACR Appropriateness Criteria for Pre-Irradiation Evaluation and Management of Brain Metastasis. 2005.).

(71)-MDR:

There may be instances other than sarcoma, melanoma, or small cell lung cancer for which initial staging may include CNS imaging. Although this may be reasonable depending on the grade, extent, and location of the primary tumor, these cases require secondary medical review.

(72)-DEF:
Melanoma is a malignant tumor of melanocytes, which are found predominantly in skin.

(73)
The incidence of melanoma is increasing more rapidly than any other malignancies, at a rate of > 4% per year (Jemal et al., CA Cancer J Clin 2008; 58(2): 71-96). Although the precise pathogenic etiology of melanoma is unknown, risk factors such as association with intermittent, intense sun exposure, older age, and exposure to pesticides have been identified (MacKie et al., Ann Oncol 2009; 20 Suppl 6: vi1-7). A family history of melanoma increases a patient's risk for developing melanoma.

(74)
CT or MRI may be performed as part of initial staging in patients with known small cell carcinoma of the lung to facilitate decision-making regarding therapy. Patients with documented CNS involvement at presentation are not candidates for prophylactic radiation therapy. Also, conventional chemotherapy is ineffective for treatment of small cell carcinoma of the lung once it has metastasized to the brain because the CNS is protected by the blood-brain barrier.

(75)
The interval for periodic assessment in stable patients is a matter of clinical judgment. Studies are generally performed no more frequently than every two cycles of chemotherapy.

(76)
The assessment is generally not necessary more frequently than every two cycles of chemotherapy.

(77)
Although any malignancy can metastasize, breast and lung cancer are the two most common primary sites of cancer in patients presenting with brain metastases (American College of Radiology (ACR), ACR Appropriateness Criteria for Pre-Irradiation Evaluation and Management of Brain Metastasis. 2005.). Patients with documented malignancy that develop new CNS symptoms or findings should undergo imaging to exclude the possibility of brain metastases.

(78)-RIN:
New or worsening CNS symptoms or findings in a patient with an AVM may be caused by a SAH resulting from a ruptured AVM; CT is then the preferred imaging study. For symptomatology that makes one suspect a SAH, see indication 400 in the "Computed Tomography (CT), Brain" criteria subset.

(79)-DEF:
An arteriovenous malformation (AVM) is a vascular lesion consisting of dilated feeding arteries and a core of tangled vascular loops that terminate in draining veins.

(80)
AVMs typically present between the ages of 20 and 40. Ruptured cerebral AVMs account for 1% to 2% of all strokes and 9% of cases of SAH. The most common presentation of an AVM is intracranial hemorrhage, with approximately 50% of patients presenting with hemorrhage (Bashir et al., Neurol Clin 2008; 26(4): 1099-1127, x; Choi and Gershenwald, Surg Oncol Clin N Am 2007; 16(2): 403-430). Each hemorrhagic event is associated with significant morbidity and 30% mortality (Cockroft, Stroke 2007; 38(12): 3310-3311; Al-Shahi and Warlow, Cochrane Database Syst Rev 2006; (1): CD003436; Choi and Mohr, Lancet Neurol 2005; 4(5): 299-308). The risk of recurrent hemorrhage is estimated to be 25% within the first year following the initial hemorrhage (Khatri et al., Neurol Clin 2009; 27(1): 109-137, viii).

(81)
Follow-up imaging is generally not indicated in stable AVM without changes in symptoms or treatment plan.

(82)
Whether to perform CT or MRI in this setting is a matter of clinical judgment.

(83)
It may be reasonable to obtain imaging studies in the immediate postoperative period following an intracranial procedure. These patients are often acutely ill and the determination of neurologic status can be difficult, as it is often complicated by sedating medication or cerebral edema. Clinical judgment may dictate early scanning if the patient fails to progress as expected, even if

worrisome focal neurologic signs are absent.

(84)
Symptoms and findings of CNS involvement by SLE or vasculitis are quite varied but typically include headache, mental status changes, seizure, or stroke. Focal or nonspecific neurologic findings may also accompany HIV, without concomitant opportunistic CNS infection. These diseases have characteristic imaging findings that aid in diagnosis and thereby guide therapy.

(85)
MRI is more sensitive than CT for detection of intracranial lesions caused by vasculitis.

(86)
Neurological involvement in sarcoidosis is seen in approximately 5% to 10% of cases; approximately half of these cases involve the CNS. Presenting signs and symptoms of CNS involvement include mental status changes (e.g., somnolence), seizures, and cranial nerve palsies. CT or MRI imaging may diagnose neurosarcoidosis but gadolinium-enhanced MRI is the preferred imaging study to evaluate the brain parenchyma, the spinal cord, and the meninges (Lower and Weiss, Clin Chest Med 2008; 29(3): 475-492, ix).

(87)
Multiple sclerosis (MS) is a chronic inflammatory disease of the CNS. The natural history of MS is characterized by the relapse and remission of various focal symptoms; some patients experience a chronic progressive pattern of disability (Courtney et al., Med Clin North Am 2009; 93(2): 451-476; Birnbaum, Adv Neurol 2006; 98: 111-124). Sites of autoimmune mediated demyelination cause focal neurologic impairment, which may correlate with MRI signal intensity changes within the white matter. The hallmark of MS lesions is their bright appearance on T2-weighted images in the brain; lesions are also commonly seen in the spinal cord (Simon, Radiol Clin North Am 2006; 44(1): 79-100; Bakshi et al., Neurology 2004; 63(11 Suppl 5): S3-11). The use of MRI has allowed earlier confirmation of the diagnosis, resulting in earlier medical intervention and improved management of the disease.
LP results and visual evoked potentials can suggest the diagnosis.

(88)
MRI is primarily used for the evaluation of suspected MS, as well as for following new or worsening symptoms. MRI can exclude other conditions that would account for the patient's symptoms and exam findings, can establish the presence of clinically silent lesions, and can demonstrate new lesions (Royal College of Physicians, Multiple Sclerosis. National clinical guideline for diagnosis and management in primary and secondary care. 2004, 197). CT is not indicated as a diagnostic test for suspected MS.

(89)
Functional, magnetization transfer, diffusion tensor, and spectroscopy MRI are now being used outside clinical trials as adjunctive measures for diagnosing and monitoring disease progression and treatment response (Ali and Buckle, Neurol Clin 2009; 27(1): 203-219, ix; Bakshi et al., Lancet Neurol 2008; 7(7): 615-625; Rovira and Leon, Eur J Radiol 2008; 67(3): 409-414; Fazekas et al., J Neuroimaging 2007; 17 Suppl 1: 50S-55S).

(90)
MS can present with sensory deficits, motor dysfunction, or cerebellar or brainstem dysfunction. There is usually no set pattern to the symptoms (Lublin, Neurol Clin 2005; 23(1): 1-15).

(91)
There are a number of diseases that may present in a similar manner to MS. These include acute disseminated encephalomyelitis, CNS vasculitis, migraine, tumor, sarcoidosis, Lyme disease, Sjogren's syndrome, SLE, and vitamin B_{12} deficiency (Courtney et al., Med Clin North Am 2009; 93(2): 451-476; Miller et al., Mult Scler 2008; 14(9): 1157-1174; Krupp et al., Neurology 2007; 68(16 Suppl 2): S7-12; Birnbaum, Adv Neurol 2006; 98: 111-124). Many of these diagnoses can be ruled out with laboratory testing (e.g., CBC, ANA, ESR, vitamin B_{12}, TSH).

(92)
Gadolinium contrast is used to identify any disruption of the blood-brain barrier secondary to active inflammation. The number of enhancing lesions is the most clinically relevant measure of ongoing disease activity (Simon, Radiol Clin North Am 2006; 44(1): 79-100, viii; Bakshi et al., Neurology 2004; 63(11 Suppl 5): S3-11).

(93)
MRI is considered positive for MS if the following criteria are met (Frohman et al., Neurology 2003; 61(5): 602-611; McDonald et al., Ann Neurol 2001; 50(1): 121-127; Tintore et al., AJNR Am J Neuroradiol 2000; 21(4): 702-706; Barkhof et al., Brain 1997; 120 (Pt 11): 2059-2069):

For dissemination in space, 3 out of 4 of the following are found:
• 1 gadolinium-enhancing brain or spinal cord lesion *or* 9 T2 brain or cord lesions if there is no gadolinium-enhancing lesion
• ≥ 1 infratentorial brain or cord lesion
• ≥ 1 juxtacortical lesion
• ≥ 3 periventricular lesions
For dissemination in time:
• The appearance on a subsequent MRI (≥ 3 months from the previous MRI) of a new T2 or gadolinium-enhancing lesion at a site different from the initial event
Newer criteria have been proposed that include dissemination in space of at least one T2 lesion in each of at least 2 locations: juxtacortical, periventricular, infratentorial, or spinal cord. The dissemination in time requires a new T2 lesion on a follow-up scan (Swanton et al., Lancet Neurol 2007; 6(8): 677-686). The modified criteria are highly specific (93%) and more accurate for the clinical development of MS than the McDonald criteria (Swanton et al., J Neurol Neurosurg Psychiatry 2006; 77(7): 830-833).

(94)
Patients may present with clinically isolated syndrome (CIS) or a monosymptomatic attack. These attacks last at least 24 hours and consist of symptoms of optic neuritis, brain stem syndrome (e.g., internuclear ophthalmoplegia), or spinal cord syndrome (e.g., partial transverse myelitis, hyperreflexia, decreased motor, bowel, or bladder control) (Miller et al., Mult Scler 2008; 14(9): 1157-1174; Simon et al., AJNR Am J Neuroradiol 2006; 27(2): 455-461).

(95)-DEF:
Optic neuritis is an inflammation of the optic nerve. Symptoms include pain in and around the eye, altered visual acuity (e.g., blurred vision), and altered color perception.

(96)
A higher risk for developing future demyelination is seen in young females presenting with unilateral or painful optic neuritis, along with the finding of MRI abnormalities at the time of the attack (The Optic Neuritis Study Group, Arch Neurol 2008; 65(6): 727-732; Miller et al., Mult Scler 2008; 14(9): 1157-1174; Thrower, Neurology 2007; 68(24 Suppl 4): S12-15).

(97)-DEF:
Ophthalmoplegia is paralysis of the eye muscles.

(98)-DEF:
Transverse myelitis is inflammation (leading to demyelination) involving the full diameter of the spinal cord but limited in longitudinal extent.

(99)
The risk of developing subsequent MS is highest when the patient presents with asymmetric, incomplete transverse myelitis (Miller et al., Mult Scler 2008; 14(9): 1157-1174; Thrower, Neurology 2007; 68(24 Suppl 4): S12-15).

(100)-MDR:
Because conventional MRI does not show remyelination or the pathophysiology of lesions well, there is a mismatch between symptomatology and MRI findings (Zivadinov et al., J Neurol 2008; 255 Suppl 1: 61-74). Currently, conventional MRI is not indicated for routine follow-up of patients with known MS unless the patient exhibits a clinical change. A new lesion by imaging may not reflect treatment failure but may be a manifestation of the natural history of the disease (Simon, Radiol Clin North Am 2006; 44(1): 79-100, viii; Filippi et al., Eur J Neurol 2006; 13(4): 313-325). Studies are ongoing regarding the correlation of MRI activity with relapse rate. The evidence varies as to whether MRI should be used to monitor treatment, rather than waiting for relapses and changes in clinical symptomatology (Sormani et al., Ann Neurol 2009; 65(3): 268-275). Until solid results point to the use of MRI for routine follow-up, requests for MRI without a clinical change require secondary medical review.

(101)
An acoustic neuroma is a benign neoplasm of the Schwann cells of the vestibular nerve (CN VIII). Although vertigo is the most common presenting symptom, it is often associated with tinnitus or unilateral hearing loss (Chawla and Olshaker, Med Clin North Am 2006; 90(2): 291-304).

(102)
MRI with gadolinium contrast is the gold standard imaging tool. It provides excellent soft tissue visualization, is the most sensitive for detecting acoustic neuromas, and will show all detectable neuromas (Curtin and Hirsch, Neurosurg Clin N Am 2008; 19(2): 175-205,

v).

(103)-DEF:
Tinnitus describes a subjective sense of ringing, whistling, booming, or buzzing within the ear.

(104)
The brainstem auditory evoked response is known as BAER or ABER and is almost always abnormal with an acoustic neuroma. Auditory brainstem response is less sensitive to small (< 2 cm) neuromas and the test is often nondiagnostic in patients with profound hearing loss (Cheng et al., J Otolaryngol 2003; 32(6): 394-399). This test is an appropriate screening in patients with limited symptoms (e.g., isolated vertigo, symmetric hearing loss, unilateral hearing loss explained by history) and therefore at low risk for acoustic neuroma.

(105)
A 15 to 20 db difference is common, but there is no absolute threshold.

(106)
The abnormal acoustic reflex and roll-over phenomenon are audiometric tests.

(107)
Routine follow-up is indicated for patients who have not undergone radiotherapy or surgical excision. Initial follow-up 6 mos after diagnosis, then annually is appropriate if tumor growth is ≥ 2 mm/year (Doherty and Friedman, Curr Opin Otolaryngol Head Neck Surg 2006; 14(5): 305-313).

(108)
Residual tumor is not uncommon following surgical excision or radiotherapy for acoustic neuroma (Curtin and Hirsch, Neurosurg Clin N Am 2008; 19(2): 175-205, v; Wiet et al., Otolaryngol Clin North Am 2006; 39(4): 751-762, vii). Routine follow-up for these patients is recommended to assess for residual tumor growth or recurrence. The frequency of follow-up is a matter of clinical judgment.

(109)-RIN:
These criteria refer to the subacute onset of vertigo. The abrupt onset of severe vertigo with associated neurologic symptoms and findings particularly in patients with risk factors for vascular disease may be a sign of inferior cerebellar ischemia, infarction, or hemorrhage (e.g., CVA) (Kerber, Emerg Med Clin North Am 2009; 27(1): 39-50, viii; Baloh, N Engl J Med 2003; 348(11): 1027-1032). If there is concern that the symptoms represent a stroke, see indication 100 within this criteria subset.

(110)
Vestibular neuronitis is inflammation of the vestibular nerve or vestibular system, usually caused by a virus. The syndrome is characterized by vertigo, nausea, vomiting, spontaneous nystagmus, and postural instability. The symptoms are typically severe for one to two days, with gradual resolution over weeks to months. It is rare to have more than one episode of vestibular neuronitis, so other differential diagnoses should be considered. The symptoms gradually resolve and in these cases, neuroimaging is not necessary. However, if a patient fails to improve or develops worsening symptoms over time, imaging is appropriate to exclude tumor as a cause of prolonged vertigo. MRI is the imaging study of choice in this setting because CT fails to visualize the posterior fossa with sufficient accuracy (Kerber, Emerg Med Clin North Am 2009; 27(1): 39-50, viii; Baloh, N Engl J Med 2003; 348(11): 1027-1032).

(111)
In a patient with vestibular neuronitis, a positive head thrust test is often seen (the eyes have to make saccades, rapid, jerky movements to refixate on a target) (Kerber, Emerg Med Clin North Am 2009; 27(1): 39-50, viii).

(112)
A wide array of medication is available to treat vertigo. Therapeutic effects are usually noted within hours of administration, so prolonged therapeutic trials are usually unnecessary.

(113)
Treatment with oral corticosteroids in the first ten days of the onset of vestibular neuronitis may shorten the course of the illness (Kerber, Emerg Med Clin North Am 2009; 27(1): 39-50, viii; Chawla and Olshaker, Med Clin North Am 2006; 90(2): 291-304). (Strupp et al., N Engl J Med 2004; 351(4): 354-361).

(114)
Antihistamines effective for vertigo include meclizine and promethazine.

(115)

Neuroleptics effective for vertigo include prochlorperazine.

(116)
Benzodiazepines can be effective for vertigo, and any member of the class is reasonable to try (e.g., diazepam, lorazepam).

(117)
Scopolamine is the main anticholinergic agent used for vertigo and is available as a transdermal patch.

(118)-DEF:
Dementia is defined as a documented decline in multiple cognitive functions (e.g., memory, language, visuospatial ability) which is severe enough to interfere with daily life.

(119)
Chronic mental status changes can have a number of etiologies, including metabolic disturbance, substance dependence, vitamin deficiency, and hereditary disease. Dementia is a specific type of chronic mental status change, without diminished awareness or altered consciousness. Prior to imaging, a thorough metabolic evaluation is indicated to assess for reversible or treatable etiologies. Although treatment of these disorders may not completely reverse cognitive dysfunction, they should be routinely screened for, and if present, treated. Depression and medication side effects also need to be excluded during the evaluation (Holsinger et al., JAMA 2007; 297(21): 2391-2404; National Institute for Health and Clinical Excellence (NICE), Dementia: supporting people with dementia and their carers in health and social care. Clinical guideline 42. 2006, 56 p.; Connelly and James, Int J Geriatr Psychiatry 2006; 21(1): 14-16). Imaging can exclude potentially treatable causes of dementia (e.g., subdural hematoma, brain tumor, normal pressure hydrocephalus) (Gallucci et al., Radiol Clin North Am 2008; 46(4): 799-817, vii; Whitwell and Jack, Neurol Clin 2007; 25(3): 843-857, viii).

(120)
The American Academy of Neurology (AAN) and European Federation of Neurological Societies (EFNS) guidelines recommend that either a noncontrast CT or MRI scan be used for the initial evaluation of patients with dementia (Waldemar et al., Eur J Neurol 2007; 14(1): e1-26; Knopman et al., Neurology 2001; 56(9): 1143-1153). MRI is the preferred imaging study, however, as it has increased specificity, improved tissue contrast, and the ability to detect focal temporal lobe abnormalities, subcortical vascular changes, and can identify different patterns of atrophy. CT is primarily used to exclude other conditions that may be amenable to treatment, (e.g. brain tumors, subdural hematomas, hydrocephalus).

(121)
Cognitive assessment in patients with mental status changes and suspected dementia should include examination of attention and concentration, orientation, short- and long-term memory, language, and executive functioning. The Mini Mental Status Examination (MMSE) is commonly used, however, there are many alternate cognitive testing instruments available. Formal neuropsychological testing provides valuable information in the cognitive assessment of a patient with suspected dementia (Waldemar et al., Eur J Neurol 2007; 14(1): e1-26; National Institute for Health and Clinical Excellence (NICE), Dementia: supporting people with dementia and their carers in health and social care. Clinical guideline 42. 2006, 56 p.; Connelly and James, Int J Geriatr Psychiatry 2006; 21(1): 14-16). Cognitive testing should include assessing for behavioral and psychological symptoms that are common in dementias and Alzheimer's disease, such as apathy, agitation, and anxiety (Waldemar et al., Eur J Neurol 2007; 14(1): e1-26).

(122)-DEF:
Memory loss is the inability to remember people, places, objects, or events from the recent or distant past. In patients with dementia, memory loss is progressive in nature and interferes with IADLs and ADLs.

(123)
Confusion is characterized by fluctuating levels of consciousness and attention. Most commonly, confusion has a toxic or metabolic etiology, but imaging may be required to exclude a structural cause.

(124)
Behavioral disturbances may reflect frontal lobe dysfunction.

(125)
Depression is often under diagnosed, especially in the elderly, and may be mistaken for dementia (and vice versa) as a cause of mental status change.

(126)
Hypothyroidism can be associated with impaired memory and psychomotor skills, which may improve after thyroid hormone replacement (Devdhar et al., Endocrinol Metab Clin North Am 2007; 36(3): 595-615, v).

(127)
Liver failure can cause hepatic encephalopathy and resultant mental status changes. Serum ammonia is a commonly used marker, which correlates with the severity of this condition; however, there is no absolute value which defines a particular stage of encephalopathy (Munoz, Med Clin North Am 2008; 92(4): 795-812, viii). The PCP should consider this diagnostic possibility prior to seeking neurologic consultation.

(128)
All medications, including nonprescription drugs and herbal supplements, should be reviewed for potential side effects and interactions. Particular attention should be paid to beta blockers, antihistamines, benzodiazepines, psychotropics, anticholinergics, opiates, and centrally acting antihypertensives.

(129)
Cerebral venous thrombosis (CVT) is an occlusion of the cerebral veins and can lead to infarction of the involved cerebral territories. This disorder may present as a slowly progressive process or as an acute neurologic emergency. Risk factors for the development of cerebral venous thrombosis include dehydration, systemic and local infection, head trauma, pregnancy, puerperium, cancer, trauma, and coagulopathies (Stam, N Engl J Med 2005; 352(17): 1791-1798). The clinical presentation of CVT is often nonspecific. Common presenting symptoms include recent onset, progressive headache frequently associated with focal neurologic deficits, seizures, and altered consciousness. A syndrome of intracranial hypertension that includes headache and papilledema can account for up to 40% of cases (Poon et al., AJR Am J Roentgenol 2007; 189(6 Suppl): S64-75).

(130)
Since the clinical presentation may be variable, cerebral venous thrombosis can be difficult to diagnose. Early diagnosis is essential for prompt appropriate treatment and improved patient outcome. Magnetic resonance venography, a component of MRA to evaluate the venous circulation, provides more definitive information when performed in conjunction with MRI. MRI and magnetic resonance venography are appropriate when cerebral venous thrombosis is suspected. CT may be used as an initial screening tool to rule out other acute cerebrovascular disorders. CT venography is emerging as a competing technique for imaging of the cerebral venous system (Poon et al., AJR Am J Roentgenol 2007; 189(6 Suppl): S64-75; Khandelwal et al., AJR Am J Roentgenol 2006; 187(6): 1637-1643; Stam, N Engl J Med 2005; 352(17): 1791-1798).

(131)
A hypercoagulable state can develop due to malignancy, sickle cell disease, coagulation disorders (e.g., protein C or S deficiency, antithrombin III deficiency), during pregnancy or the postpartum period, or from the use of oral contraceptives.

(132)
A mass arising from the skull (e.g., calvarium) can result in occlusion of the cerebral veins.

(133)-DEF:
Normal pressure hydrocephalus is an acquired hydrocephalus involving impaired CSF reabsorption. The pressure of the CSF usually remains within normal range.

(134)
The classic triad of symptoms of normal pressure hydrocephalus is urinary incontinence, apraxic gait, and dementia. Gait and balance difficulties often appear before urinary incontinence and cognitive decline (Factora and Luciano, Clin Geriatr Med 2006; 22(3): 645-657; Wilson and Williams, Clin Geriatr Med 2006; 22(4): 935-951, viii). Sometimes only one or two symptoms are present.

(135)
CT or MRI findings, which suggest normal pressure hydrocephalus, include enlarged ventricles with intact cerebral parenchyma; the latter distinguishes this disorder from Alzheimer's disease where cortical atrophy is significant (Gallia et al., Nat Clin Pract Neurol 2006; 2(7): 375-381; Wilson and Williams, Clin Geriatr Med 2006; 22(4): 935-951, viii).

(136)-DEF:
An apraxic gait is slow, shuffling, short-stepped, and broad-based. It results from the inability to preplan proper foot and leg positions, or translate that plan into motor patterns.

(137)
In patients with known normal pressure hydrocephalus and new or worsening CNS symptoms or findings, a CSF shunt may be needed to improve symptoms. Imaging is helpful in assessing the size of the ventricles and helping to determine the need for shunt placement.

(138)
Obstructive (or tension) hydrocephalus develops when there is obstruction to the flow of CSF, creating an accumulation of fluid in the ventricles and thus increased ICP. It can result from intraventricular bleeding, SAH, tumor, or infection of the brain and spinal cord. Symptoms include headaches, papilledema, temporal lobe seizures, CSF rhinorrhea (the leakage of spinal fluid from the nose), and mental status changes (Goetz, Textbook of clinical neurology, 3rd ed. ed. 2007, xvii, 1364 p.).

(139)-DEF:
Ataxia is incoordination or awkwardness in performance of a motor task. The term ataxia is often used to describe gait unsteadiness.

(140)
Impaired coordination suggests cerebellar pathology. Inability to perform rapid alternating movements, finger to nose testing, or heel to shin testing are examples of impaired coordination.

(141)-DEF:
Huntington's chorea is a dominantly inherited neurodegenerative disorder that characteristically becomes manifest in midlife. Typically patients develop personality and behavioral changes, choreic movements, and slowly progressive dementia.

(142)
MRI abnormalities associated with Huntington's chorea in patients with moderate disability include atrophy of the putamen and caudate nucleus; the MRI may be normal in patients in the early stages of the disease. If MRI is nondiagnostic or normal, PET or SPECT may show metabolic abnormalities or changes in the striatal and extra-striatal regions, often before the onset of disabling symptoms (Harris et al., Med Clin North Am 2009; 93(2): 371-388; Montoya et al., J Psychiatry Neurosci 2006; 31(1): 21-29).

(143)
The diagnosis of Huntington's chorea can be made by genetic testing. If genetic testing cannot be performed because it is unavailable or the patient refuses testing, imaging is appropriate.

(144)
Progressive ataxia may be caused by a variety of neurodegenerative disorders including MS, Friedreich's ataxia, idiopathic sporadic cerebellar ataxia, tumors of the posterior fossa, and paraneoplastic cerebellar degeneration. While MRI provides excellent visualization of plaques typical of multiple sclerosis (MS) and may diagnose posterior fossa tumors, the differentiation of the remaining etiologies may be more difficult. If the MRI is nondiagnostic, PET may be helpful, as it will show hypometabolism in the cerebellum and brainstem for idiopathic sporadic cerebellar ataxia and will show hypermetabolism in the same areas for Friedreich's ataxia (Gilman et al., Neurology 2008; 71(9): 670-676).

(145)
MRI is a component of the preoperative assessment for these procedures because it can be difficult to locate appropriate anatomical targets for deep brain stimulation or lesion creation in patients with structural abnormalities seen on MRI (e.g., cortical atrophy, ventricular enlargement) (Koepp and Woermann, Lancet Neurol 2005; 4(1): 42-53; Camacho and Castillo, Semin Ultrasound CT MR 2007; 28(6): 424-436).

(146)
Subdural hematomas develop as the result of trauma. In acute cases, there is often a clear history of head trauma with a rapid progression of symptoms that may lead to coma. In chronic cases, the head trauma may have been minor and even forgotten. Symptoms (e.g., slowness in thinking, confusion, apathy, drowsiness) may not become apparent for a period of weeks following the precipitating event (Bullock et al., Neurosurgery 2006; 58(3 Suppl): S16-24; discussion Si-iv; Karnath, Geriatrics 2004; 59(7): 18-23).

(147)-DEF:
Bradyphrenia is slowness of thought and information processing.

(148)
Pupillary changes include asymmetry and an abnormal reaction to light.

(149)
Advanced age is associated with a greater risk of falls resulting in traumatic injuries, and a greater likelihood of medical conditions which require treatment with anticoagulation therapy. Other factors, including age-related cortical atrophy, increase the risk of chronic subdural hematoma in the elderly. In addition, primarily because of these age-related changes, the clinical presentation of the disease in the elderly varies from the presentation in younger adults. A common clinical presentation in the older patient includes headache, altered mental status, hemiparesis, gait disturbance, and aphasia. Higher rates of mortality in the elderly from chronic subdural

hematoma have been attributed to this variation in clinical presentation resulting in a delay in diagnosis (Karnath, Geriatrics 2004; 59(7): 18-23).

(150)

A subdural hematoma occurring following CSF shunting for normal pressure hydrocephalus is considered a mechanical failure (Chen and Levy, Neurosurg Clin N Am 2000; 11(3): 399-406).

(151)

CT is the preferred study for the initial evaluation of suspected subdural hematoma (Bullock et al., Neurosurgery 2006; 58(3 Suppl): S16-24; discussion Si-iv). MR imaging is more sensitive than CT in the detection of small subdural hematomas and subdural hematomas that are in the subacute phase (Provenzale, Emerg Radiol 2007; 14(1): 1-12).